Medical Societies

ONTARIO MEDICAL ASSOCIATION

THE thirty-fifth annual meeting of the Ontario Medical Association was held in Peterborough May 25th to May 28th, 1915, in conjunction with the annual meeting of the Medical Officers of Health. On Tuesday, May 25th, the fourth annual conference of the Medical Officers of Health of the province of Ontario was opened under the presidency of Dr. W. R. Hall, of Chatham. The attendance was good, about 280 members being present. At the morning and afternoon sessions several interesting papers were read, and in the evening the Mayor of Peterborough gave an address of welcome. The question of salaries for medical officers of health was discussed at the afternoon session on Wednesday and a committee appointed to consider the matter suggested the following schedule: townships, $300 per annum; incorporated villages up to 1,000 population, $150 per annum, with addition of $50 for each additional 1,000 population or majority thereof; towns, $100 for first 1000 population with the addition of $50 for each subsequent 1,000 population; cities under 20,000 population $1,000 per annum; cities of larger population $1,200 per annum. This schedule was adopted. It was pointed out that, in the case of towns and cities, the remuneration given should be sufficient to enable the medical officer to devote his whole time to the duties entailed by his position as medical officer of health. The election of officers resulted as follows: president, Dr. A. W. McPherson, of Peterborough, who is now on active service; vice-president, Dr. A. J. Macauley, of Brockville; secretary-treasurer, Dr. J. W. S. McCullough, of Toronto; committee on papers, Dr. Roberts, of Guelph, Dr. F. A. Dales, of Stouffville, and Dr. J. W. S. McCullough, of Toronto.

On Wednesday afternoon, a business meeting of the Ontario Medical Association took place, the president, Dr. D. J. Gibb Wishart, in the chair.

After the reading of the minutes, the following were elected to the nominating committee: Drs. H. J. Hamilton, J. H. Elliott, W. A. Young, A. Moorehead, F. C. Harrison, J. Loudon, N. D. Buchannan, H. B. Anderson, and W. H. B. Aikins. The following reports were then received and adopted: The Treasurer's, by J. H. Elliott; Audit by J. M. McColloch and E. A. Hammond; Credentials by

W. K. Colbeck; Papers and Business by H. J. Hamilton. At 4 p.m. the City of Peterborough and the local profession entertained the Association by an excursion over the Lift Locks, afterwards serving refreshments in the Armouries. The evening session met in the Collegiate Institute. The programme consisted of the President's address (see page 469 of the JOURNAL) and a paper on the clinical manifestations of syphilis, by Dr. J. G. Phillips, of Cleveland.

Thursday morning was taken up with the meetings of the sections in Medicine, Surgery, Obstetrics and Gynæcology, and Ear, Eye, Nose and Throat. In the afternoon, Professor F. J. Shepherd of Montreal delivered the address in Surgery; Dr. A. H. Wright gave a paper on Medical Education and Fee-Splitting, and Dr. A. F. McKenzie one on the local Medical Society. In the business meeting which followed, the Secretary read a report of the committee on the affiliation of the County Medical Societies which recommended the following:

1. "That a member should belong to a local Society before becoming a member of the Ontario Medical Association, special provision being made for admission for members living in districts not yet organized.

2. "Membership in a local Society makes a member eligible for the Ontario Medical Association and his fee may be collected through the local Society.

3. "It shall be the duty of this committee to obtain the volunteer services of members of the Association to aid the organization of societies."

Dr. H. J. Hamilton presented the report of the Committee on the relations of the Ontario Medical Association and the Canadian Medical Association, reporting progress and asking leave to sit again. After some discussion these reports were adopted.

Resolutions:

Moved by Dr. Bruce, seconded by Dr. H. J. Hamilton, that the Association have a paid organizer for the coming year. Carried.

Moved by Dr. C. Myers, seconded by Dr. Hamilton, "That this Association desires to place itself on record in favour of active steps being taken immediately for the prevention of insanity by the establishment of separate neurological wards in general hospitals, especially in those hospitals in which clinical teaching is given." Carried.

The Nominating Committee then presented its report:

Place of meeting, Toronto; President, Dr. H. B. Anderson; vice-presidents, Drs. G. S. Cameron, Peterborough; A. T. Emmerson,

Goderich, C. B. Oliver, Fort William; W. K. Colbeck, Welland; general secretary, Dr. F. Arnold Clarkson; assistant secretary, Dr. F. C. Harrison; treasurer, Dr. J. H. Elliott; delegates to the Canadian Medical Association, Drs. D. J. Gibb Wishart, F. C. Neal, and H. J. Hamilton.

Moved by Dr. J. H. Elliott, seconded by Dr. W. A. Young, that the usual honorarium be paid the secretaries. Carried.

Moved by Dr. Elliott, seconded by Dr. F. N. G. Starr, "That we, the members of the Ontario Medical Association, assembled in anuual meeting wish to express our deep appreciation of the loyalty and self-sacrifice of our fellow members of the profession who have offered themselves and are now serving the Empire and its allies at the front in the various services. In thus serving the cause of freedom, many will come back under conditions which will practically mean beginning their professional life over again. As fellow practitioners, we wish to assure them that as far as lies in our power, we will see that their old clientèle awaits them on their return to civil practice, and that their relations to former patients will as far as possible be restored. For the fulfilment of their arduous duties, we wish them health and strength, and we express our sincere hopes of a speedy and safe return to their homes and families." Carried.

The following resolution was passed unanimously by the section of Obstetrics and Gynæcology: "That the general session appoint a committee to consider and enforce adequate measures for a campaign amongst the members of the medical profession and the public to guard against the ravages of cancer, by early diagnosis and treatment; and to report at the next meeting of the Ontario Medical Association." Carried.

Moved by Dr. Colbeck, seconded by Dr. N. D. Buchanan, "Be it resolved that this Association deplores the fact that very inadequate provision has been made for the payment of medical and hospital fees of workmen injured in the industrial concerns of the province. While some manufacturers, in addition to their contribution to the funds of the Compensation Board, have made ample provision, many others have done nothing whatever. We would ask that the Compensation Board take steps in co-operation with the Government, to have amendments made to the Workingman's Compensation Act, so that workmen, who received only half pay while incapacitated, will not be under the necessity as at present, of paying these charges out of their already too-scanty earnings." Carried.

Moved by Dr. A. A. McDonald, seconded by Dr. H. B. Anderson,

that a hearty vote of thanks be tendered to the Mayor and Council of the City of Peterboro, and to the Ladies' Committee, for their great kindness and hospitality to the members of this Association, and that a copy of this resolution be sent to the Mayor. Carried.

The new officers were then installed.

The evening session was held in the Collegiate Institute.

On Friday morning sectional meetings were held in the Armouries. The total registration was 176.

MONTREAL MEDICO-CHIRURGICAL SOCIETY

THE ninth regular meeting of the Society was held Friday February 5th, 1915, the President, Dr. W. F. Hamilton, in the Chair.

PATHOLOGICAL SPECIMENS: Series by Horst Oertel, M.D.

1. *Large liver abscess of obscure origin and etiology.* The patient, a man thirty years of age, entered the Royal Victoria Hospital apparently suffering from an acute infectious disease, which originally was diagnosed as probable typhoid fever. This, however, was soon questioned. He presented more or less indefinite abdominal symptoms and, at first slight, and later more accentuated, pulmonary symptoms, pointing to the presence of fluid in both chests and rather indefinitely to cavitation. The Widal reaction was negative, white blood cell count 17,600, temperature varied from 98° to 104·2° with a pulse of from 76 to 140. Urine concentrated with a slight amount of albumen, no casts. The disease ran in a general way the course of a septicæmia.

Autopsy disclosed double exudative fibrinous pleurisy, both lungs floating in a large amount of turbid fluid. The lungs showed multiple abscesses. The right lung contained four or five intercommunicating small abscesses in the middle lobe, with similar ones in the lower and upper part of the upper lobe. Some were also in the lower lobe near the base. There existed a certain amount of purulent infiltration of the lung tissue immediately surrounding these abscesses. The left lung showed only one or two small abscesses in its upper lobe. There was nowhere extensive pneumonic consolidation, save that limited purulent infiltration of lung tissue surrounding the just-mentioned intercommunicating, apparently metastatic, abscesses.

Searching for other foci, attention was immediately directed to the tremendously enlarged liver, which had pushed the dome of the diaphragm on the right side upward, although the liver itself was

quite free from it and not adherent. On the left side, the liver had been pushed by this enlargement of the right lobe upward and backward, so as to reach the dome of the diaphragm on the left side and resting against the spine. However, it was everywhere free from adhesions. The structures of the portal spaces were free, portal glands not prominent. The portal vein and the vena cava inferior contained fluid blood and were otherwise unchanged. The enlargement of the liver was almost entirely at the expense of the right lobe, the upper part of which was extremely bulging and on percussion fluctuated. This enlargement of the right lobe was responsible for the displacement of the rest of the liver substance to the left, upward and backward. The weight on removal was 4,075 gm. On section a tremendous amount of creamy, greenish, but non-odorous pus escaped from the upper part of the right lobe and it was found that the largest part of the upper lobe, leaving only about 7 cm. of the right lower lobe, was transformed into intercommunicating abscess cavities. The whole of the right lobe of the liver measured, after removal of the pus, 25 × 17 cm. The left lobe to the interlobar notch measured 10 × 15 cm. The left lobe was flat and apparently quite uninvolved. A further examination of the iliac veins and the inferior vena cava, as well as the carotid arteries and the aorta, showed these vessels only filled by fluid blood. The mesenteric glands were not prominent. The stomach, duodenum, gall-bladder and ducts were free. Nothing of any further importance was discovered at autopsy and section had been limited to abdomen and thorax, so that other structures in this regard important, as, for instance, the middle ear, could not be examined. Under the circumstances, it is not easy to trace the origin of the lesion and determine its etiological factor.

In the first place, the bacteriological examinations, kindly made by Dr. Bruère and Dr. Mason, were interesting, but not conclusive. During life, the pleural fluid was found sterile and non-pathogenic to mice and guinea pigs. At autopsy, some of the pus from the liver abscess and some from the lung was examined with the following results: In the lung were predominatingly hæmolytic and non-hæmolytic streptococci, two diphtheroid bacilli, a few definite pneumococci and organisms of very similar morphology to those which were practically pure in the liver abscess. These latter were gram-positive diplococci growing in staphylocoid masses. They were not capsulated and were impossible to cultivate ærobically and anærobically on media containing blood serum. This organism was found non-pathogenic for mice and guinea pigs. The

bacteriological examination, therefore, leaves the etiology rather misty.

Tracing now the origin, it appears from examination of the liver, particularly where the lesion is still more recent, that the extension within the liver corresponds to what is usually characteristic of a portal infection. As is well known, infections of the liver may enter through the bile ducts, the lymphatics, both of which could be easily ruled out in this case, or the vascular system. Of these ports of infection, the venous system is, of course, the most important. This may be concerned either through the portal system or by retrograde infection through the hepatic veins. It was, as will have been seen, perfectly impossible to trace any portal infection in this case. There existed no ulceration in the gut, mesenteric arteries and veins were perfectly intact and, as previously recorded, all the structures of the portal spaces were free. The question arises, therefore, whether it is possible in this instance to deal with an arterial infection or one through the medium of the hepatic veins. Now, infection through the hepatic artery rarely produces extensive large abscesses in the liver. It is most frequently encountered in septic endocarditis and then usually produces small miliary abscesses, but this does not correspond at all to what we have here before us. There remains, then, the possibility of infection through the hepatic veins by retrograde embolic metastases from a distant focus. In middle ear disease it is, for instance, possible that bacterial emboli may reach the jugular vein, the superior vena cava, the right auricle; travel backward through the inferior vena cava to the hepatic veins, thence to the liver, where they primarily form small embolic abscesses. But these, in breaking down, may involve, and then extend within, the portal system of the liver, thus gradually simulating a true portal infection. In other words, the picture of a portal infection in the liver may result from a general bacteriæmia, and I am inclined to believe that this is what occurred in this case, although original focus and port of entrance remain obscure. As to the direct etiological factor, we are, of course, unable to make any statement with any definiteness, but there is some foundation,—not much, but some,—that the case belongs to the category of pneumococcic pyæmias. What bacteriological evidence we possess, further the character of the pus, and, finally, past experience are in its favour. It is certainly most peculiar and interesting that this diplococcus stained exceedingly well but resisted all attempts at cultivation and had lost all its pathogenic power. If it, therefore, belongs to the group of pneumococci,

or, indeed, under any circumstances, it is certainly a very attenuated organism which must have lost its pathogenic property in the develo ɔment of the disease.

2. *Carcinoma of cervix extending to parametrium.* This second specimen is an extensive carcinoma of the cervix, inoperable at the time of admission to the Royal Victoria Hospital, which illustrates exceedingly well the progress of this disease. The specimen shows the cervix absolutely destroyed, necrotic, in process of cavitation; but one also sees that growth and ulceration stop abruptly at the body of the uterus. The disease does not extend upward beyond the cervix, but sideways into the right parametrium, producing a similar extensive ulcerative growth. Within the parametrium it has travelled upward so as to involve the right ovary and right tube, leaving the body of the uterus intact. Thus the progress of this carcinoma is not one by continuity or even contiguity, but by preference typical of lymphatic extension. The case was complicated by involvement of the ureters, leading to their dilatation and an early hydronephrosis.

3. *Complete tuberculosis of both supra-renal glands, communicating with vein on left side, miliary tuberculosis, no Addison's disease.* In a case of miliary tuberculosis of both lungs, complicated by tuberculous meningitis, was found a very advanced cheesy tuberculosis of both supra-renals. In the left super-renal practically no normal tissue remained. Sections of what might grossly still be regarded as a remnant of supra-renal tissue in the upper pole, show even this transformed into tuberculous cheesy masses. A larger vein is found to communicate directly with the cheesy masses in the centre of the gland, so that, undoubtedly, a ready access was established to the rest of the circulation. The right supra-renal was not quite so diffusely affected, but nodular masses also permeate it very generally. This case is not only interesting as illustrating a rare origin of the miliary tuberculosis, but because symptoms and signs of Addison's disease were lacking entirely. In this connexion, however, one anatomical peculiarity of the case is interesting: the surroundings of both supra-renals were free. As is well known, immediately beneath them lies the solar plexus with a rich sympathetic system. In the majority of cases of tuberculosis of the supra-renals, associated with Asddion's disease, they are found to involve, by adhesion or extension, the underlying sympathetic system. Whether this absence had anything to do with a lack of development of Addison's syndrome in this case can, of course, not be definitely stated, but a record of this observation seems worth while. Earlier ob-

servers of Addison's disease, as, for instance, Virchow, were inclined to attribute much greater significance to the involvement of the neighbouring sympathetic structures than to the supra-renal itself, and Virchow stated that other lesions in the situation, as inflammations or growths from the stomach or glands might be associated with Addison's syndrome, if they affected this sympathetic plexus. Later investigators have, however, denied this and attributed the disease to an interference with the internal secretion of the supra-renal gland. It seems difficult to see how any secretion could have been continued in this case. No other, accessory, supra-renal glands were found at autopsy. One must, therefore, suppose that other parts of the chromaffinic system compensated.

DEMONSTRATION of lantern slides illustrating diseases of the auricle with electro-cardiograph charts, by Dr. J. C Meakins.

DISCUSSION: Dr. T. F. Cotton: These slides of Dr. Meakins are of particular interest to me, and I am sure to all of us who are interested in heart work. The newer methods of examining the heart tell us a great deal about the significance and nature of disorders of the auricle and the important rôle they play in producing heart failure. Perhaps there is no alteration in the mechanism of the heart that is of more interest to the clinician than great acceleration of the auricular rate, both as an explanation of the symptomatology and as an indication for treatment with drugs of the digitalis group. As Dr. Meakins has shown, there is a very close relationship between auricular flutter, premature auricular contractions, and auricular fibrillation. In many cases the onset of the auricular flutter is preceded by individual isolated premature auricular contractions for a period of months. A large number of cases of auricular flutter, certainly the majority of those which we treat, terminate in fibrillation. The symptoms of auricular flutter depend not so much upon the auricular tachycardia as upon the great increase in the ventricular rate. Auricular flutter may be present in a heart that is apparently healthy, yet that heart, if the paroxysm be of sufficient duration, will eventually suffer from the great work put upon it and dilatation and venous stasis are not rare sequelæ. There will be a quick return to normal in the healthy heart with the resumption of the normal rhythm. It is, however, quite different with the diseased heart; one with mitral stenosis, for instance; this is a heart which has, perhaps, already lost a great deal if not all of its reserve and it cannot support the burden that has been put upon it and heart failure may result. There are three things which we

may hope to do in these cases, with digitalis, when given in large doses; we may either bring on auricular fibrillation which is temporary and which is followed by a normal rhythm, or the fibrillations may remain permanent, or we may be forced to give digitalis for a long time and not succeed in more than reducing to within normal limits the ventricular rate. Those cases in which fibrillation remains permanent certainly are improved subjectively; they lose that sensation of oppression in the chest and that feeling of fluttering in the region of the heart. In those cases in which we only succeed in increasing the heart block we can prolong the life of the patient for many years.

The case which Dr. Oertel has shown is particularly interesting in association with the last slide Dr. Meakins presented. I remember a case of complete heart-block under Dr. Finley, with Stokes-Adams syndrome extending over a year. With each fit for three minutes there would be no heart sound and no pulse, then the heart would start to beat and then a high-tension pulse of 120. Each time following a fit it would be 120 and gradually within five minutes would slow down again to its normal rate of 32. The slide which Dr. Meakins showed, giving a series of ventricular premature contractions almost presenting tachycardia, was most interesting. My case showed a similar electric curve, a complete heart block, the same dissociation of the auricles and ventricles, and these ventricular premature contractions occurring in succession were the cause of the increase in the pulse rate. The patient died and post mortem examination revealed a large heart with considerable myocardial degeneration. The heart was lost and section could not be made.

PAPER: The use of salvarsan in pernicious anæmia, by Dr. H. A. Lafleur.

DISCUSSION: Dr. C. F. Martin: We are all much indebted to Dr. Lafleur for his very lucid description of the cases he has treated by this method and we are all struck by the fair proportion of cases that have been improved by it. The experience of a good many has been not quite so satisfactory. It seems to me, as I noted the blood count, that none, or comparatively few, if any, have a very high blood count even when most successfully treated; the hæmoglobin rarely rose above 50 per cent. and comparatively few of the red cells rose above three million. However, the subjective improvement seems to be so very marked in some that Dr. Lafleur is to be congratulated. In a few cases in which we have used it in the Royal Victoria Hospital we were not so fortunate in our results. In one

case there was a slight increase in temperature and rigours and the patient went from bad to worse and succumbed to the disease under the clinical picture of septicæmia. A great deal of improvement has followed this treatment, probably more than by any other, and yet I think cases sometimes have improved quite as remarkably by other means; such, for example, as saline injection, transfusion, and sometimes even by the much despised cacodylate of sodium. I think there are a great many cases which will naturally get well for a time no matter what the treatment and although these cases may not be classed as cured they certainly show a very marked improvement.

Dr. M. Lautermann: As one who has had occasion to use a great deal of salvarsan during the past five years, Dr. Lafleur's contribution to-night presents many points of interest to me. It is gratifying to know that the sphere of usefulness for this arsenical preparation is extending. I should like to ask Dr. Lafleur if a Wassermann reaction was made in each one of his cases to exclude lues; some writers have held that pernicious anæmia is often due to latent or remote lues and I have felt myself that it probably might be well to be sure on this point in the treatment of the condition. Another factor that occurred to me, particularly in the second case where the neo-salvarsan had been used, while in the other eleven cases the old salvarsan proper was employed; it occurred to me that neo-salvarsan, being less toxic might possibly have been better tolerated. I have treated three cases of pernicious anæmia with neo-salvarsan, one case of my own and two brought to me by a confrère. The first is still alive and after four years is very comfortable, and one may be justified in regarding this as being cured; the other two cases were both improved, one treated ten months ago is still doing fairly well and still taking arsenic by mouth, the other was lost sight of. I do not know that we can claim for salvarsan or neo-salvarsan, any more than for arsenic, that it will bring about a cure, but there is one thing we can claim for it and that is much more rapid action than any other preparations with which we are familiar, and it does not appear to lose its efficiency when used in relapses, which frequently occur.

Dr. H. A. Lafleur: Of course I sedulously avoided using the word "cured" in this connexion. All I wanted to maintain is that having treated cases of pernicious anæmia for some time, and using various things, I have never seen results quite so good as this. I have treated cases with Fowler's solution and, to begin with, the majority of people do not stand large doses. I hope Dr. Martin

understood that I was not speaking of cure in these cases and I specially laid stress on the fact that I was not by any means going entirely by the blood picture. On the contrary many patients feel very well with a lower blood picture than those with a high one, and they must be really in as good condition. After all, subjective sensations must count for something, and one's capacity for living a more or less normal life. I am just really experimenting with salvarsan and I am quite pleased with it so far; and I have not had other results that are at all comparable to these. As to the Wassermann reaction I think I stated that in some of the reports it was positive and in others negative, but whether the test was done in all the cases I cannot say. As far as neo-salvarsan is concerned, I only used it in case No. 2, and one would expect that this was quite as efficient as the other preparations.

CASE REPORTS: 1. Two cases of diaphragmatic hernia, Dr. J. Stowers.

2. Case of relapsing fever in a laboratory boy who contracted the disease by allowing a tick, which had been brought here from South Africa, to bite him. Dr. J. Stowers.

THE tenth regular meeting of the Society was held Friday evening February 19th, 1915, in the Montreal General Hospital. The members of the Society were the guests of the Medical Board of the Hospital and the following programme was arranged by a committee appointed by the board, of which Dr. E. M. von Eberts was chairman.

PART I (Surgical Theatre): A case of enlarged spleen in a young child; regarded as an early stage of Gaucher's disease, Dr. Blackader. The differential diagnosis between tic and chorea, Dr. Shirres. A diphtheria carrier, Dr. A. H. Gordon. Subacute bacterial endocarditis, Dr. Lafleur. The new department of remedial gymnastics, Dr. Harvey. B. typhosus osteomyelitis, Dr. Hutchison. Nail extension for fracture of femur, Dr. Elder. Case of appendicular gastralgia, Dr. Bazin. Case—Carcinoma of ampulla of Vater; excision; recovery, Dr. von Eberts. Acute hæmatogenous nephritis, Dr. Patch. Decompression for fracture of skull, Dr. Henry; *X*-rays of stomach following gastro-enterostomy, Dr. Wilkins. Vincent's angina, Dr. Hamilton. Fracture of nose; *x*-ray, Dr. Craig.

PART II (Governors' Hall) Exhibits: Typhoid and paratyphoid fever charts, Dr. Lafleur. *X*-ray plates, Dr. Wilkins. Pathological specimens, gross and microscopical, Dr. Rhea. Photo-

graphs of skin diseases, Dr. G. G. Campbell. Apparatus for intravenous anæsthesia, Dr. Hepburn.

Demonstrations: Pathological specimen, Epidermoid carcinoma hand, Dr. Bazin. Laryngeal tumour, Dr. Hamilton. Laryngeal tumour, Dr. Craig. Case—Bone graft for the relief of Talipes equino varus, Dr. Nutter.

PART III (Eye Section): Demonstration in small surgical room of a series of living cases, chiefly congenital abnormalities, Dr. Mathewson.

PART IV: Demonstration of the electro-cardiograph.

Dr. W. Grant Stewart called the meeting to order and Dr. J. Alex Hutchinson was chairman for the evening.

At the conclusion of the programme, coffee and sandwiches were served in the Governors' Hall.

THE eleventh regular meeting of the Society was held Friday March 5th, 1915, Dr. W. F. Hamilton, president, in the chair.

Dr. A. D. Blackader presented before the Society some notes he had made of the New British Pharmacopœia.

CASE REPORTS: 1. Primary syphilis of the conjuctiva. 2. Fibroma of the orbit, by Dr. G. H. Mathewson.

Dr. L. J. Rhea showed a slide from the fibroma case.

PAPERS: 1. Some observations on eclampsia, by Dr. T. F. Cotton. This paper was discussed by Dr. Horst Oertel and Dr. H. M. Little.

2. Back ache, corsets, and high heels, by Dr. J. Appleton Nutter, illustrated by charts.

THE twelfth regular meeting of the society was held Friday, March 19th, 1915, the president, Dr. W. F. Hamilton, in the chair.

PATHOLOGICAL SPECIMENS: Series by Dr. Horst Oertel

1. *Unusual aortic and pulmonary ulcerative endocarditis with double perforation.* This specimen is from a patient who was in the Royal Victoria Hospital, under the care of Dr. Hamilton and particularly studied by Dr. M. E. Abbott, with a clinical diagnosis of malignant endocarditis. The case will be reported in detail later by Dr. Abbott. Suspicion was entertained during life of the patient that a communication existed between the right and left heart. At autopsy was found an ulcerative and productive endocarditis, extraordinary in position and extent. It involved the pulmonary and aortic valves with their adjoining endocardium. The endocarditis was ulcerative in so far as it had led to great de-

struction of both valves; productive, as it had caused the formation of multiple large polypoid vegetations on the valvular cusps which, by continuity and contiguity had extended to the parietal endocardium on both sides. This is more pronounced on the right side. The vegetations are at least partly organized and are firmly attached to the valves and parietal endocardium. The interest in this case is heightened by the fact that there are two communications between the right and left sides; one from the sinus of Valsalva of the middle cusp of the aortic valve by an aneurismal pouch which is directed towards, and has perforated into, the right ventricle immediately below the pulmonary valve; the second communication is established through the musculature of the interventricular septum immediately below and between septal and middle cusps of the aortic valve by an irregular ulcerated canal. In this regard it is interesting and important to enquire into the possibility of previous developmental cardiac anomalies which might have determined the seat and extension of the lesion. For it is known, of course, that cases of malignant endocarditis may, as their point of predeliction, occupy, particularly, septal cardiac defects. Without going into details as to the complicated development of the heart, we may recall here shortly that heart anomalies occur generally as the result of either of two development faults; first, failure in fusion of primary septa; second, irregular and faulty development of septal limbs. The latter occurs especially through certain inhibitory actions which allow parts of an advancing septum to develop more rapidly than others, thereby destroying developmental continuity. This may happen in the interventricular septum, which divides the ventricles and which develops by a dorsal and ventral limb, the former growing, even under normal conditions, more rapidly than the ventral one. Consequently developmental communications occur almost entirely at the point of fusion of septa, or, occasionally, within the course of a septum. But in this heart neither communication between the right and left ventricle is in a situation which may readily be accounted or by such developmental faults. On the contrary, it appears that development of the septa as well as their fusion and the division of aortic trunk into aorta and pulmonary artery have been quite well performed. If this point is against a developmental communication between left and right heart, we possess, in addition, the anatomical evidences of the perforations themselves. The one is a definite aneurismal pouch whose walls and orifices are beset with necrotic vegetations. The other is a ragged irregular canal

with no evidence of a previous definite communication. It is surrounded by thickened endocardium and vegetations. In view of these facts it appears that the weight of evidence points to a purely ulcerative double perforation between the two sides.

2. *Extensive congenital hydrocephalus in a child of seven months.* The patient was under the care of Dr. Archibald. On the death of the child Dr. Archibald immediately injected both carotids with 10 per cent. formalin, with the result that the specimen was found at autopsy, some hours afterwards, in an extremely good state of preservation. It is quite typical in general character. The tremendously dilated lateral ventricles practically control the whole brain area and this dilatation extends well into the anterior and posterior horns. It is interesting to note that the grey matter of the cortex is relatively better preserved than the white matter. Of the occipital lobe, particularly, all that can be seen is a thin covering, made of flattened convolutions of grey matter with no white substance at all. The basal ganglia are nearly entirely lacking and only anteriorly a part of what appears to be the corpus striatum, over which hangs the choroid plexus, exists. The anterior part of the much dilated third ventricle shows the anterior commissure in the form of a thin thread. The duct between the third and forth ventricle is short and wide and there is no obliteration of the spinal canal. This specimen is interesting because it throws some light upon a disputed point in the pathogenesis of congenital hydrocephalus; namely, whether it is due to obstruction or obliteration within the ventricles or ducts of the cerebro-spinal canal. In this case there was certainly no evidence that any such obstruction, either of developmental or inflammatory origin, existed anywhere. It seems also questionable to me whether cases of congenital hydrocephalus can only be attributed, as has frequently been done, to hyper-secretion of cerebro-spinal fluid. It seems at least plausible to assume that, in addition, a lack of development of brain substance is concomitant and possibly precedes it and that the tremendous increase in fluid may at least partly be accounted for by the lack of normal advance in development of brain substance.

3. *Amyloid degeneration of liver, spleen and kidneys following Pott's disease.* Presentation of a typical "sago" spleen; a liver with a rather diffuse amyloid degeneration, which, however, was not massive but only involved extensively the capillaries; and a kidney with amyloid degeneration of the glomeruli, following a case of Pott's disease and hip-joint tuberculosis.

4. *An unusual splenomegaly.* This extraordinary spleen, which will form the subject of a later detailed publication, was an accidental finding in a recent autopsy of a man who had died shortly after an accident. There is practically no history to this case. The only knowledge we have is that he was struck on the perineum and scrotum by an iron bar while at work, which immediately was followed by great pain and marked swelling of the parts. The following day he was admitted to the Royal Victoria Hospital. The findings then were: male of forty-two years, fairly well nourished with markedly swollen scrotum, size of a grape-fruit, deep purple in colour. Right groin also swollen and discolured as far as the anterior superior spine of the ileum. Of his previous history we only know that he had pleurisy on the right side three years ago. His condition continued fairly good, but on the sixth day after admission evidences of an infection were found which were relieved by multiple incisions in the right groin and scrotum and drains inserted. During the following night he had considerable hæmorrhage and developed rapidly the symptoms of a general septicæmia. He died during the evening of the seventh day after admission.

At autopsy the tissues of the scrotum were found œdematous and gangrenous and the adjoining perineum equally so. There existed also a hæmatoma of the abdominal wall extending from the pubis to about 2 cm. below the umbilicus. The heart showed a marked concentric hypertrophy of the left ventricle. The right pleura showed adhesions between the lobes, but was clean over the surface. On the surface of the lower lobe of the left lung there were a few minute tubercles, and near the base there was a larger calcified, slightly contracted, superficial area, but the apex was free. A very unusual condition, however, was found with regard to the spleen. This occupied almost completely the left hyperchondrium and was adherent by dense old fibrous adhesions to all the surrounding organs and structures. Above it was bound firmly to the diaphragm, on the side to the stomach and tail of pancreas and below to the kidney and supra-renals. On removal it was found very markedly enlarged (weight 1225 gm., measurement 12 × 12 × 9 cm.). The surface showed deep scar contractions, dividing it into irregular lobes and giving the organ somewhat the appearance of the lobated liver in syphilitic cirrhosis, while its surface, however, was not irregular, granular or nodular. The organ as a whole was firm and on section it cut firmly, leaving a smooth surface which presents a very characteristic mottled appearance due to diffuse

irregular markings on a pale greyish ground substance. These markings are made up of reddish streaks, dots and blotches. In addition to these there occur, particularly at the periphery, fibrous scars, corresponding largely to the interlobar depressions and extending into the spleen as irregular trabeculae. These traverse the spleen in the form of whitish hyaline bands which occasionally show a yellowish discolouration. They often surround and follow the course of blood vessels within the splenic substance so that their walls are surrounded by thick yellowish white scar tissue. Where these vessels have been cut in longitudinal section, they may be followed in the form of threads or short bands. Occasionally such blood vessels occur within the previously described hæmorrhagic blotches and streaks. It will be seen from this description that we are dealing with an unusual type of splenomegaly and it is not easy to decide what etiological factor may be responsible for it and how to classify it under the existing categories of splenic enlargement. That it is not due to the septic condition, in other words, that it is not an acute splenic hyperplasia, seems quite certain, but it also does not fit in with the chronic types of splenic enlargement which occur in chronic infections or in diseases of the blood. The microscopic examination, which, of course, has not been completed, seems also to exclude the unusual forms of chronic splenomegaly which are grouped under the term of Gaucher's type. There remains the infective granulomata. Whether we have to do with an unusual form of tuberculosis or syphilis or a lesion related to Banti's disease must be left to a future decision, which I hope to make when the case is reported in detail. It is unfortunate that the clinical history of the case is so incomplete and that no knowledge of blood examination during life is available. The case, aside from its scientific importance, is certainly interesting from a clinical stand-point, for it illustrates how frequently diseased conditions may exist in persons considered healthy for all practical purposes and that only an autopsy will give us all the desirable information.

Discussion: Dr. A. D. Blackader: Dr. Oertel, if I understand his remarks aright, regards the existence in chronic hydrocephalus of any obstruction of the ducts at the base of the brain, which permit fluid to escape from the lateral ventricles, as being disproved. Does he thereby exclude inflammatory processes at the base of the brain producing an occlusion of these ducts as a cause of hydrocephalus? I have always considered that chronic hydrocephalus might be due to several quite distinct conditions,

but have always regarded acute or chronic inflammations of the meninges at the base of the brain as a very definite proven cause of some cases of hydrocephalus in infancy.

Dr. H. Oertel: My remarks were made with reference to developmental or congenital hydrocephalus. I think we must, at the beginning, separate cases of hydrocephalus into two categories, namely, those which are truly developmental and those which are acquired. A considerable number of cases of hydrocephalus which occur after birth belong to the acquired type, and it is quite possible that these occur as the results of inflammatory lesions or obstructions within the ventricles or intercommunicating ducts or meninges. These lesions, however, are, as the case which I presented to-night illustrates, absent in the true developmental hydrocephalus and it, in my opinion, depends upon an inhibition in the formation of brain substance which allows the accumulation of fluid, more or less, as a compensatory process.

DEMONSTRATION: A series of fractures, illustrated by *x*-rays, By Dr. F. R. England.

CASE REPORT: Coma with acidosis, in a case of acute artnritis, by Dr. W. F. Hamilton. The patient was a young woman who had just weaned her child and the breasts were still full and active. She developed an acute arthritis and was placed under treatment with sodium salicylate when marked signs of acidosis and coma supervened. The patient recovered. (A full report of this case will appear later.)

PAPER: The paper* of the evening was read by Mr. V. H. Mottram of the Physiological Department of McGill University, on Fat metabolism including its relation to acidosis.

DISCUSSION: Dr. A. D. Blackader: The case just cited by Dr. Hamilton is one of much interest. The dose of the salicylate was not in my opinion excessive, and any tendency to the production of acidosis was well safeguarded by the amount of sodium bicarbonate combined with it. The results produced, if they were entirely due to the salicylate, were certainly very rapid in appearing and I think must be regarded as an unusual idiosyncrasy on the part of the patient. Occasionally very marked cases of idiosyncrasy to the action of this drug are reported. I would like to ask Dr. Mottram to tell us the reason of the enlargement of the liver which frequently occurs in acidosis in infancy. In my experience an amount of fat in the infant's dietary exceeding 4 per cent. is very liable to give rise to symptoms of acidosis with marked en-

*See page 603.

largement of the liver. In these cases we meet with vomiting, a peculiar rapidity of breathing, an absolute inability to take food, especially if it contain fat, and a liver, of which the lower margin may reach the level of the umbilicus. In one fatal case in which I was able to secure an autopsy Dr. McCrae stated the liver to be loaded with fat. One of the early symptoms of the development of acidosis is the ammoniacal smell which the urine in this condition develops, owing to the conversion of a portion of the urea in the system into ammonia which is then used for neutralizing the acid in the system, and is promptly eliminated in the urine. I would be glad to be informed by Mr. Mottram whether in the infant we should always regard an ammoniacal odour to the urine as an indication of an early stage of acidosis.

Dr. D. J. Evans: I have been much interested in this paper by Mr. Mottram and also in Dr. Hamilton's case; there is much that is suggestive in both of them. As to the question of the lactation in Dr. Hamilton's case; it has occurred to me that it is possible the symptoms had something to do with it. The breasts were engorged at the time of admission to the hospital and the patient may have been one of those women who are exceptionally good nurses. The urine showed that there was an absorption of lactose; may there not also have been an undue absorption of fat in the same way? If she had not been in a state of active lactation at the time, when the fat metabolism was undergoing active work, there might not have been the same results.

Dr. Mottram's paper as to the condition in pregnancy and during lactation of the fat metabolism is extremely interesting. It has always been an extraordinary thing to me to notice the great similarity in the phenomena of the acidosis of pregnancy, with what one meets in childhood; the symptoms are so similar. I have never understood how it is that women have this acid indigestion in pregnancy. The excessive demands upon the liver in this direction are probably the explanation of this form of acidosis in pregnancy that is so frequently met with. It is probably along this line where the liver breaks down under the strain, that we have the eclamptic state developing, when the condition passes over from the physiological to the pathological. The whole subject is one of great interest. These are the first steps that are being taken up just now by various observers along the lines of the metabolism of pregnancy, and in this study we find the liver and the metabolism of fat playing a very interesting rôle.

Dr. H. Oertel: I think that, in a general way, pathological

anatomists and physiologists are in agreement to-day on the subject of fatty infiltration and we all agree that even an appreciable amount of fatty infiltration may occur in an organ under perfectly normal conditions. On the other hand, I do not believe that all fatty infiltration of organs may be regarded as normal or comes within physiological limits. It is, of course, true that fatty infiltration in itself does not, strictly speaking, constitute a pathological condition of cells, but I think we must admit that excessive and permanent fatty infiltration of an organ marks more or less definitely an injury to its cells and, therefore, makes that organ pathological; for it certainly indicates that the healthy metabolism and the regenerative ability of the organ have suffered and that it keeps itself alive under difficulties with the aid of fat as an easily combustible substance. Thus we see degenerative changes, atrophy, or other nutritive disturbances in cells frequently associated with fatty infiltration, and this may be followed by return to healthy conditions or, in lasting injury, to progressive and permanent fatty infiltration. In these cases fat can be seen to infiltrate gradually the cells of the organ in the form of typical, usually large, fat drops which are deposited within the cell body, where they acquire more and more room at the expense of the protoplasm and gradually squeeze the nucleus towards the cell membrane. Now I think we must, even to-day, differentiate, as much as Virchow did formerly, this form of fatty change in organs from another which Virchow named fatty degeneration, although we hold to-day different views as to its character than he did. This type of fatty appearance in organs occurs usually in more rapid and virulent protoplasmic destructions. It does not, at least in the beginning, show the gradual large fat drop infiltration as previously outlined, but fat or physically fat similar, small, almost pin or dust-like, droplets appear diffusely, although rather irregularly, within cells whose protoplasm and nuclei show the severe qualitative changes incident to rapid disorganization of cell constituents. We interpret this lesion at present as an active cell destruction in which lipoid and cholesterin drops are released from the emulsified non-visible state within normal protopasm. It is this type of fatty change in organs which occurs within severe toxic, often rapidly fatal, lesions and which in its character and genesis seems to stand out more or less sharply from that of fat infiltration by ordinary glycerine esters. Undoubtedly both forms of fatty changes are frequently found combined, probably always in cases where death does not rapidly supervene and when restitution

to integrity takes place, or is at least attempted, in the manner which has been so well described by Mr. Mottram to-night.

Mr. V. H. Mottram: In answer to Dr. Oertel I may say that I do not think my point was that fat infiltration of *any* organ is physiological, but that fat infiltration of the liver is physiological. In no case that I have dealt with can the tissue be called in any way pathological. Histological preparations have always been made and there is no sign of damage to the nucleus, etc. I think I would go so far as to say that fat percentage in the fresh liver may vary between 2 and 22 without there being anything wrong with the liver; but as regards the heart and kidney it cannot go above 3 per cent. without there being something decidedly wrong. We have yet to settle how far a liver can function normally when there is a large amount of fat present. To judge from the literature of the subject you can have big infiltrations without serious consequences.

As regards the case which Dr. Hamilton reported I may say that I have found nothing in a recent view of the action of salicylates to lead one to expect an acetonuria. But there is undoubtedly disturbed fat metabolism in his case and the acetonuria may be not only an indication of the mobilization of fat from the mammary gland but also from the fat depôts. The temperature chart shows that the temperature of the patient went, on February 3rd, below normal and that the acetonuria then developed. I found that my animals at the end of the first day of hunger had a subnormal temperature; they then called on their reserve fat, and after that the temperature went above normal. I would like to suggest that the fall in temperature is the signal for fat mobilization, and that, in Dr. Hamilton's patient there was mobilization occurring on account of the subnormal temperature.

Regarding the point that Dr. Blackader raised, the enlargement of the liver that is seen sometimes in children, accompanied with acidosis, this may possibly be the result of a fatty infiltration. Certainly in a large number of animals I have seen, when there has been an excessive amount of fat in the liver, the liver looks yellow and swollen, feels firm, and tears in a quite different way from that of glycogenic liver.

As regards the ammonia in urine in fat metabolism and acidosis I think it is pretty clear that wherever there is an acidosis you get an excess of ammonia. The two run parallel. The explanation that physiologists give is that it is not ammonia formed *ad hoc*, but ammonia caught on its way to conversion to urea. It is linked with the acid and excreted as such.